ILLUMINATING GAS POISONING.

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Chemistry. The employment of gas manufactured from coal for illuminating purposes is over a century old. The original method was by the destructive distillation of coal or wood in retorts, with subsequent purification. Ammonium and sulphur compounds are the most objectionable impurities of crude gas, and much attention has been given to their removal. The gas-product by this method is principally hydrogen, hydrocarbons, and a very small amount (7 per cent.) carbon monoxide. Toxic qualities of illuminating gas produced in this manner are very low. In recent years the trades, to reduce the cost and simplify the production, have developed new methods of forcing steam through hot coal or coke. The action is not distillation, but oxidation, of coal to carbon monoxide, which is itself reduced by hydrogen. To this "water gas" is added hydrocarbons, that is, methane, ethane, benzene, etc., to fit it for illuminating purposes. The most striking characteristic of "water gas" is its toxicity, due to the excess of carbon monoxide, of which gas it contains 45 per cent., or 38 per cent. more than the carbon monoxide content of coal gas. Carbon monoxide possesses a strong chemical affinity for hemoglobin, with which it combines to form carbon-monoxide-hæmoglobin, resisting decomposition by free oxygen. Hufner states that 1 gram of hemoglobin will take up 1.3 c.c. of carbon monoxide.

Causes. The causes of illuminating gas poisoning are:

1. Hygienic: Deficiency of instruction as to its use, defects of construction of apparatus, and deficiency of ventilation.
2. Psychopathic: Insanities, acute and chronic, exciting absorption of illuminating gas into the blood by inhalation.

Hygienic causes necessarily are responsible for those cases of poisoning which are accidental (non-suicidal), and usually result from lack of instruction of those inexperienced in the manipulation of gas-lighting fixtures. Most cases of poisoning by illuminating gas, however, are the result of suicidal premeditation.

Occurrence. The number of cases of illuminating gas poisoning occurring in large cities has been progressively on the increase in the last ten years. This is certainly the reverse of our expectations, and is a medicosociological question worthy of solution. It cannot be attributed to defects of plumbing, as our buildings are more carefully and intelligently inspected by municipal officers today than they were ten years ago. The use of illuminating gas has been diminishing as electricity succeeds it for lighting purposes.

The enormous increase in the number of cases of poisoning by illuminating gas may be attributed to several changes in the conditions of men: (1) Concentration of population in cities, and (2) increased susceptibility to emotional states and insanity. The enactment of rigid laws restricting the sale of toxic drugs to the layman has also had a tendency toward the increase of poisoning by illuminating gas. The following statistics, while somewhat indefinite, are valuable as an indication of the extent of increase of illuminating gas poisoning:

Deaths from Poisonous Gases in New York City.

1867–1880 (twelve years)	16
1880–1892 (twelve years)	202
1889	37
1890	54
1891	64
1902	353
1906	419

Philadelphia.

1899	30
1900	19
1901	26
1902	36
1903	45
1906	50

These are deaths known to be suicidal. In 1906 in Philadelphia 74 deaths from inhalation of poisonous gases were reported in addition to the 50 known to be suicidal. In Boston 43 fatal cases were reported in 1904.

City.	Deaths from inhalations of poisonous gases, 1906.	Suicides by asphyxia, 1906.
San Francisco, Cal.	35	24
Denver, Col	2	3
Washington, D. C.	14	12
Atlanta, Ga.	3	1
Chicago, Ill.	126	55
New Orleans, La.	4	1
Baltimore, Md.	14	9
Boston, Mass.	18	10
Kansas City, Mo.	10	2
Buffalo, N.Y.	5	1
New York, N.Y.	250	169
Cincinnati, Ohio.	6	1
Cleveland, Ohio	6	1
Philadelphia, Pa.	74	50
Pittsburg, Pa.	5	...
Galveston, Tex.	3	...
Norfolk, Va.	3	1
Richmond, Va.	4	...

Comparative studies of these figures from many points of view may be interesting and instructive, but it is not my object to treat of the subject statistically. Having presented the figures, I wish to justtify my study of the clinical picture by the progressive increase in death rate. Suffice it to say that in nineteen cities approximately 4000 cases of poisoning from gases, with 582 deaths, have occurred; 340 of the cases were suicidal.

SYMPTOMATOLOGY. The symptoms of gas poisoning are the manifestations of the abolished interchange of oxygen for carbon monoxide in the alveoli of the lungs, and the absence of oxidation of the detritus of cell energy in the body, together with the combination of the constituents of illuminating gases with the physiological elements, changing them into toxic substances. The characteristic features of acute gas narcosis are not constant, and the severity and duration of toxic symptoms depend largely upon the duration of exposure.

Respiratory System. Much has been written of the effect of illuminating gas on the pulmonary system. The respirations are rapid and labored—often of the Cheyne-Stokes type; the face is cyanosed. There is often mouth frothing, the froth frequently containing blood. Most of the fatal cases present, sooner or later, pulmonary œdema as a complication, which is considered the cause of the incident rise in temperature and leukocytosis. I have seen cases in which there was no evidence of pulmonary œdema, with rise in temperature and leukocytosis. W. Gilman Thompson has found œdema, bronchopneumonia, acute congestion, emphysema, pleurisy, and atelectasis at necropsy.

Cardiovascular System. The pulse in all cases is rapid, feeble, and perhaps irregular, being out of all proportion to the temperature and respiration. Examination of the blood shows a most conspicuous change. In a series of cases studied during my incumbency in the Emergency Hospital in 1904 and 1905, I found the red cells reduced 3,000,000 to 1,000,000, and the hemoglobin from 14 per cent. to 69 per cent., the degree of reduction being inversely proportional to the duration of exposure and severity of symptoms, The leukocytes were increased, varying between 9000 and 22,000. Morphologically, the red blood cells showed marked poikilocytosis, granular and hydropic degeneration, polychromatophilia, and enucleation. Polychromatophilia is especially conspicuous, and would seem to confirm Engel's theory, that these cells appear when rapid new formation of red corpuscles is demanded and when there is not time for complete shrinkage of nucleus and development of orthochromatic cells. The color index was minus, and the specific gravity and coagulability increased. Thompson reports an increase in red blood cells, but my study of 15 cases, herewith appended, fails to corroborate his observations except in the early stages (two to six hours), when the red cells and hemoglobin were usually normal or slightly increased. The anemia was, in favorable cases, followed by a return to normal; but in fatal cases the anemia persisted or increased. While there was a marked change in the hemocytic elements, probably due to effects of carbon monoxide, I am of the opinion that the primary increase in red blood cells and leukocytes is in a large measure due to the transudation of blood serum from the vessel walls into the tissues. The whole blood is increased in specific gravity and coagulability, with the presence of non-inflammatory œdema of the lungs, cutaneous blebs, and often serous leptomeningitis. The polycythemia, if due to a decrease in the volume of the plasma, is only a relative polycythemia, and the concentration of solid elements of the blood only temporary. Much importance was attached to the primary increase in red cells per cubic millimeter during the early stages, and the phenomenal decrease after a few hours. The pronounced secondary anemia occurring within five or six hours after the onset of coma is in a degree due to the fact that most of these cases are submitted to venesection and intravenous transfusion of $\frac{N}{10}$ saline solution, which produces hydremia and low red cell count. The presence of abnormal morphology, with slow return to normal is in favor of hemolytic anemia. The blood pressure is at first increased, but after prolonged exposure rapidly diminishes.

			Temperature.				Blood findings.		
	Exposure.	Coma.	Maximum.	Minimum.	Pulse.	Respiration.	Leukocytes.	Red blood corpuscles.	Hemoglobin.
1.	2 hours	3 hours	100°	98°	112	30	10,000	5,000,000	80%
2.	3 hours	5 hours	101°	98.4°	118	30	No rep't		
3.	2 hours	1 hour	100°	98.2°	126	28	No rep't		
4.	3 hours	9 hours	100.4°	98°	126	30	12,000	4,600,000	70%
5.	½ hour	None	99.4°	98°	100	24	9,000	4,000,000	80%
6.	5 hours	72 hours	108°	100.8°	160	68	18,000	3,000,000	20%
7.	8 hours	12 hours	105°	96.8°	140	48	No rep't		
8.	3 hours	10 hours	100.8°	98°	120	32	No rep't		
9.	6 hours	24 hours	106.6°	103.2°	140	48	No rep't		
10.	Unknown	38 hours	108°	98.2°	164	66	22,000	4,200,000	30%
11.	6 hours	60 hours	101°	97.4°	132	62	No rep't		
12.	4 hours	16 hours	103°	99°	112	40	13,800	......	66%
13.	Unknown	4 hours	99°	97.8°	100	22	No rep't		
14.	4 hours	12 hours	99.2°	97°	100	26	12,300	4,612,000	49%
15.	Unknown	18 hours	100.4°	97°	100	40	15,000	4,200,000	50%

Nervous System. Coma is prompt after short exposure to illuminating gas. The reflexes are usually completely abolished. The pupils are usually contracted and do not react, but may be dilated, normal, irregular, or respond to light. Temporary nystagmus is common. Muscular twitchings may precede or follow coma.

Most cases present incontinence of the sphincters. Delirium is a usual complication. Hemetson reports a case with convulsions lasting nine days. Among diseases of the nervous system following gas poisoning, the following have been reported: hemiplegia, dementia, acute mania, serous leptomeningitis, disseminated encephalitis, multiple sclerosis, and multiple neuritis.

Urinary System. The urinary findings are for the most part negative. In three of my cases the urine showed evidence of an acute hemorrhagic nephritis.

Skin. The skin is usually cyanosed, cold, and dry. I wish to call attention especially to the appearance of blebs on the extremities and back, usually occurring in chains, in cases in which the coma persists for twelve hours or longer. These bullæ contain clear serum, and have frequently been mistaken for the effects of excessive external heat applied in treatment. They were present in five cases, all of which ended fatally.

Temperature. The temperature is usually subnormal during the first twelve hours, and reaction is characterized by a rapid rise, varying from 99° to 103° F. The temperature returns as rapidly to normal as it reached the fastigium.

Vagaries of temperature are due to: Disturbances of normal chemical processes; disturbance of circulatory equilibrium, or altered pulse and respiration.

In cases in which exposure has been prolonged, and in which coma is consequently deep, the temperature rises rapidly, reaching 104° to 110° F. These cases terminate fatally within twenty-four to seventy-two hours. The rise in temperature is usually coincident with the development of œdema, but I do not agree that the sudden development of high fever is caused by the pulmonary condition, but rather incline to the view that it is the result of overwhelming toxemia, the œdema being of hemolytic origin.

DIAGNOSIS. The history of the case is sufficient to make the physician reasonably certain of the diagnosis, but the fact that a patient is found in coma in a room stifling with the odors of illuminating gas without any mode of free entrance of atmospheric air should not be sufficient to forego a careful examination for evidence of other toxic substances introduced by the suicide to make the termination certain. At least a small percentage of the cases of illuminating gas poisoning are complicated by the effects of toxic doses of other poisons capable of producing death.

PROGNOSIS. The prognosis should be based upon a careful and intelligent consideration of the following facts: Duration of exposure; degree of coma; condition of the pulse, respiration, and temperature; condition of lungs; condition of the blood; and the presence of cutaneous blebs. If the exposure be long, the coma is correspondingly deepened and more prolonged. Coma for forty-eight or more hours augurs unfavorably. The onset and rapid development of œdema with pronounced hemolytic changes is unfavorable. The persistence of anemia and coma with the onset of trophic changes should be looked upon as indicating a most doubtful result. Short exposure to illuminating gas is followed by prompt response to treatment, with recovery. All the cases which develop cutaneous blebs end fatally.

The cause of death is not, in all cases, asphyxia. The leukocytosis, hemolysis, frequency of complications involving the nervous system, and constant pathological changes in the brain and spinal cord, strongly indicate toxemia.

PATHOLOGY. Little was known of the morbid anatomy until the investigations of Thompson in 1904. The most marked changes are found in the blood. The kidneys frequently show evidences of acute nephritis of the glomerular type, and many of the patients who live for twenty-four hours or more undoubtedly succumb to an intercurrent uremia. The lungs may show no changes whatever; however, œdema, acute congestion, and emphysema are common in the cases coming to autopsy. Changes in the brain and spinal cord are by far the most constant pathological findings. Parenchymatous neuritis, multiple sclerosis, serous leptomeningitis, encephalomyelitis, and cerebral hemorrhage have been reported.

TREATMENT. The management of patients suffering from intoxication by illuminating gas has been unsatisfactory, because of the affinity of CO for hemoglobin, and lack of knowledge of the pathological and metabolic processes which the intoxication produces. Inhalation of oxygen has always been unjustly esteemed in the treatment. The use of oxygen by inhalation is of doubtful efficacy, since it seems improbable that the excess of oxygen over that in normal atmosphere can become physiologically combined with the hemoglobin in exchange for carbon products.

The patient should be taken from the room and into fresh air. The tongue should be retracted from the mouth and so retained by means of a hemostat, improvised gag, or tongue retractor. If necessary, artificial respiration should be begun at once. The patient should be removed without delay to an institution or physician's office, where immediate phlebotomy should be performed. A pint to a pint and a half of blood should be removed, and simultaneously a quart of $\frac{N}{10}$ saline solution transfused in the median basilic or cephalic vein of the opposite forearm. Venesection can be repeated two hours after the first bloodletting, if the patient be not doing well. Saline solution should be given subcutaneously every two hours in quantities of one pint, or by the rectum continuously. Saline solution diminishes toxemia, lessens the tendency to œdema of the lungs, increases the affinity of red cells for oxygen, and stimulates the circulatory system. At the outset the patient should be given by hypodermic injection ether, 30 minims, atropine, $\frac{1}{100}$ grain, and suprarenalin, 30 minims. Nitroglycerin and vasodilators in general should be avoided. Suprarenalin is administered on a rational basis, presuming the œdema, etc., to be the result of increased capillary permeability. The patient should be protected from exposure by an abundance of blankets. External heat should be applied (bearing in mind that the skin is especially susceptible to heat).

While venesection and intravenous injection of saline solution are being done, an assistant should proceed to wash the stomach thoroughly with a solution of potassium permanganate, and follow lavage by the introduction of magnesium sulphate (3 ounces). The respiration and pulse should be watched closely, and stimulated carefully as indicated. Urinalysis should be made daily.

Crile has advocated direct transfusion of blood, and has recently reported results of animal experiments, but his statistics do not show a greater percentage of recoveries than do the active stimulation, venesection, and intravenous injection of saline solution.

Deductions. Death from gas poisoning is rapidly on the increase; changes in the blood are conspicuous and severe; toxemia is pronounced; the severity of the symptoms and the duration of the coma depend upon the duration of the exposure; complications of the nervous system are common; cutaneous vesicles appear frequently in severe cases; elevations of temperature occur independently of œdema; poisoning by illuminating gas is sometimes complicated by toxic symptoms of other poisons; saline solution diminishes œdema of the lungs; oxygen is useless; direct transfusion is impracticable; asphyxia is not often the cause of death; the bloodvessels are dilated; the tendency to exudation of serum is increased; suprarenalin in repeated doses is indicated.

CIRCUMSCRIBED SEROUS SPINAL MENINGITIS.[1]

A LITTLE-RECOGNIZED CONDITION AMENABLE TO SURGICAL TREATMENT.

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A collection of clear fluid in a cyst of the spinal pia-arachnoid is a condition little known in America, as only one case occurring in this country is on record (Spiller, Musser, and Martin,

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[1] Read at a meeting of the College of Physicians of Philadelphia, December 2, 1908.